Amendments to the Specification:

Please delete paragraph [0098], which starts with "Figures 10-B (SEQ ID NO: 3)".

Please replace paragraph [0099] with the following amended paragraph:

[0099] Figures ~~11A-B~~ 10A-B show the functional properties of serial deletions of RAP-2. In Figure ~~11A~~ 10A, there is a schematic representation of the consecutive C-terminal deletions of RAP-2. All truncations share the intact RAP-2 N-terminus, while their C-terminal ends are designated by arrowheads. The RIP, NIK, IKKß and TIP60 binding region is underlined. Three hatched boxes correspond to the putative leucine-zipper-like motifs. Figure ~~11B~~ 10B shows the effect of overexpression of the deletion constructs described in A on NF-κB activation in HEK-293T cells by RelA, TRAF2 TNF and NIK using the HIV-LTR luciferase reporter plasmid for NF-κB. Activation of the reporter gene luciferase activity is expressed in Relative Luciferase Units (R.L.U.).

Please replace paragraph [0100] with the following amended paragraph:

[0100] Figures ~~12A-B~~ 11A-B show mapping of RAP-2 functional and binding regions.

Please replace paragraph [0101] with the following amended paragraph:

[0101] In Figure ~~12A~~ 11A, various deletions of RAP-2 were tested for their ability to bind the indicated proteins within transfected yeast (odd columns) and mammalian HEK-293T

cells (even columns). The two rightmost columns show the ability of the same deletions transfected at high amounts as detailed in example 9 into HEK-293T cells, to inhibit NF-κB activation and potentiate c-Jun hyperphosphorylation (c-Jun) in response to TNF-α treatment. Boldness of the crosses is proportional to the intensity of a given effect. Asterisks indicate that the observed effects of the labeled constructs towards Rel-A stimulation are distinct (see Figure ~~11B~~ 10B).

Please replace paragraph [0102] with the following amended paragraph:

[0102] (Figure ~~12B~~ 11B is a summary of the chart representing localization of the binding (upper part) and functional (bottom part) regions of RAP-2 as inferred from the deletion analysis shown in Figure ~~12A~~ 11A, aligned along the protein backbone. The hatched parts indicate possible location of borders of the corresponding minimal regions.

Please replace paragraph [0103] with the following amended paragraph:

[0103] Figure ~~13~~ 12 shows that ser-148 in RAP-2 is essential for its ability to induce c-Jun hyper phosphorylation at ser-63.

Please replace paragraph [0240] with the following amended paragraph:

[0240] RAP-2 becomes phosphorylated when RAP-2-IKK1 complex, immunoprecipitated from transfected HEK293 cells, is incubated under *in vitro* phosphorylation conditions. A search for the functional role of the phosphorylation of RAP-2 revealed

that mutation of one particular serine in this protein (in position 148) fully abolishes the activation of Jun phosphorylation by it. As illustrated in Fig. ~~13~~ 12, while overexpression of the wild type RAP-2 resulted in a massive increase in Jun phosphosylation, overexpression of RAP2 (S148A) did not affect at all the phosphorylation of Jun. The effect of RAP2 on NF-κB, however, was not affected at all by this mutation. These findings indicate that phosphorylation of serine 148 in RAP2 is specifically involved in its effect on Jun phosphorylation.

Please replace paragraph [0243] with the following amended paragraph:

[0243] Applying the full-length RAP-2 protein as bait in two-hybrid screen of a B-cell cDNA library, we have isolated a novel protein interacting with RAP-2 denoted hereafter clone #10 or clone #10-encoded protein or RAT-binding protein #10 or RBP-10 ~~(Figure 10)~~ (SEQ ID NO:3). The original clone (about 2.2 kb) was found to encode a putative polypeptide of apparent MW of 60kDa. However, the putative ATG first codon is apparently missing from this sequence. Despite its considerable length, the obtained cDNA has therefore to be expanded further towards the 5' end to reconstitute the entire open reading frame.

Please replace paragraph [0251] with the following amended paragraph:

[0251] By employing consecutive deletion analysis, the binding regions within RAP-2 were mapped and RIP, NIK, TIP60-

binding as well as the self-association domain(s) were identified (Figure ~~11~~ 10A).

Please replace paragraph [0253] with the following amended paragraph:

[0253] So far neither the IKKβ nor the NIK binding sites (amino acids 95-264) and (amino acids 1-264) respectively were found to overlap RIP's binding site within RAP-2 (Figure ~~11~~ 10A).

Please replace paragraph [0256] with the following amended paragraph:

[0256] The protein encoded by clone #10, with the above-mentioned exception, apparently binds within a region beginning between amino acids 218-309 and ending at amino acid 416 and thus, its binding site may comprise overlapping regions with the binding sites for RIP, NIK, IKKß and TIP60 (Figure ~~11~~ 10A).

Please replace paragraph [0257] with the following amended paragraph:

[0257] To the extent of our present knowledge, all the functional effects of RAP-2 (namely NF-κB inhibition and induction of c-Jun hyper-phosphorylation) map to the same region (Figure ~~11~~ 10B).

Please replace paragraph [0260] with the following amended paragraph:

[0260] Moreover, with the exception of RelA, the effect of all inducers used in our experiments can be mediated by as few as approximately 100 N-terminal amino acids of RAP-2. In fact

even the fragment encompassing amino acids 1-102 mediates a distinct effect, albeit fairly moderate (Figure ~~12B~~ 11B).

Please replace paragraph [0262] with the following amended paragraph:

[0262] From the results shown in Figures ~~11 and 12~~ 10B and 11A, it appears that: